Submicron particle-bound polycyclic aromatic hydrocarbons in the Polish teaching rooms: Concentrations, origin and health hazard

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ABSTRACT

The goal of the work was to investigate the concentrations of the 16 US EPA priority polycyclic aromatic hydrocarbons (PAH) bound to submicrometer particles (particulate matter, PM_1) suspended in the air of university teaching rooms and in the atmospheric air outside. Two teaching rooms were selected in two Polish cities, Gliwice, southern Poland, and Warsaw, central Poland, differing with regard to the ambient concentrations and major sources of PM and PAH. The variabilities of indoor and outdoor 24-hr concentrations of PM_1-bound PAH, the ratio (I/O) of the indoor to outdoor 24-hr concentrations of PM_1-bound PAH, probable sources of PAH and the level of the hazard from the mixture of the 16 PAH (ΣPAH) to humans at both sites were analyzed. In both Warsaw and Gliwice, the mean concentrations of PM_1-bound ΣPAH were slightly higher in the atmospheric air than in the rooms. The indoor and outdoor concentrations of individual PAH in Gliwice were correlated, in Warsaw – they were not. Most probably, the lack of the correlations in Warsaw was due to the existence of an unidentified indoor source of gaseous PAH enriching PM_1 in phenanthrene, fluorene, and pyrene. Although the ambient concentrations of PM_1-bound PAH were low compared to the ones observed earlier at both sites, they were much higher than in other urbanized European areas. However, because of low mass share of heavy PAH in ΣPAH, the various indicators of the health hazard from the 16 PAH mixture were low compared to other regions.

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Introduction

In general, the air quality inside buildings depends on the atmospheric air quality. This dependency can be questionable for hermetically sealed rooms, where air is exchanged through ventilation or air conditioning systems, but if there are neither forced air exchange nor inside pollution sources, even air tight doors and windows do not prevent the pollutant concentrations in the room and in the atmospheric air outside from being very close. In hot climate zones, where the windows and doors are

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permanently open, this dependency is obvious, even when inside pollution sources exist (Jones, 1999; Monn, 2001; Morawska and Salthammer, 2003).

The closed doors and windows in normal buildings practically are not an obstacle to ambient fine particulate matter (fine PM: $PM_{2.5}$, PM_1) getting inside (Jones et al., 2000; Morawska and Salthammer, 2003). In public facilities (schools, nurseries, offices, libraries), if there are no indoor pollution sources, the indoor fine PM concentrations are close to (or slightly higher than) the PM concentrations outside. (e.g., Hassanvand et al., 2015; Romagnoli et al., 2014; Oliveira et al., 2016; Krugly et al., 2014; Zhu et al., 2015; Alves et al., 2014; Błaszczyk et al., 2017). However, because polycyclic aromatic hydrocarbons (PAH) adhere to PM particles only in some specific conditions, the indoor concentrations of PM-bound PAH can be visibly lower than outdoor ones (ibid.). The mass proportions of the PM-bound and volatile phases of PAH, especially of 2-, 3-, and 4-ring ones, are not stable (Tobiszewski and Namieśnik, 2012; Keyte et al., 2013). They vary with varying ambient concentration and chemical composition of PM, ambient concentrations of co-occurring substances, temperature and relative humidity of air, etc. (Ravindra et al., 2008; Rybak and Olejniczak, 2014).

PAH are considered one of the most hazardous components of PM. They are the first air pollutants identified as carcinogens by the US Environmental Protection Agency (US EPA). The strength of their carcinogenicity grows with their molecular weight. After entering the human body, PAH are conveyed by blood to various organs, where they are metabolized (Nikolao et al., 1984; White, 2002; Keyte et al., 2013). The metabolites, after they covalently bond with DNA or RNA, cause neoplasms, affect replications, transcriptions, and biosynthesis of proteins (Skupińska et al., 2009). All PAH can accumulate in the body tissues. However, it is unknown so far if the carcinogenicity of PAH, which in the air occur always as a mixture, never as a single compound, may be ascribed to individual hydrocarbons or if it is due to the concerted effects of some number of PAH. The carcinogenicity of a PM-bound PAH may be enhanced or suppressed by other PM components.

PM_1, the core part of PM in urbanized areas, contains majority of PM-bound PAH (e.g., Rogula-Kozłowska, 2015, 2016). In Poland, especially in the urbanized areas of its southern part, ambient concentrations of PM- and PM_1-bound PAH are highest in Europe (Rogula-Kozłowska et al., 2013; Rogula-Kozłowska, 2015). The indicator of PM-bound PAH air pollution, PM_{10}-bound benzo(a)pyrene (BaP), has been routinely monitored in Western Europe since 2005 (the limit for its yearly concentrations is 1 ng/m³). In Poland, in 2015, the highest yearly PM_{10}-bound BaP concentrations reached 15.6 ng/m³ at some sites in southern and central Poland, and their average over 137 measuring sites scattered over the country (117 urban background, 7 suburban, 8 regional background, 3 traffic sites, and 2 sites affected by industrial emissions) was 5 ng/m³ (GIOŚ, 2016). Practically, neither indoor concentrations of PM-bound PAH (including BaP) nor the effects of outdoor on indoor PM-bound PAH concentrations have been investigated in Poland so far.

The US EPA recognizes sixteen PM-bound PAH as priority pollutants, and seven of them as carcinogens (Wang et al., 2010). The goal of the present study is to investigate the concentrations of PM_1-bound phase of these 16 PAH in two university teaching rooms and their dependence on the outdoor PM_1-bound PAH concentrations. Teaching rooms are usually smaller than, for example, public libraries, museums, theaters, etc., and more intensely used (many people, usually during the better part of a day). The PM samples were taken in two Polish cities, Warsaw in central Poland, and Gliwice in southern Poland, differing with the structure of the PM emissions. Probable sources of PAH and the level of the health hazard to humans from the mixture of PAH (i.e., equivalents: carcinogenic, CEQ, mutagenic, MEQ, and TCDD-toxic (TCDD - 2,3,7,8-Tetrachlorodibenzo-p-dioxin), TEQ) were also determined.

1. Material and methods

The PM_1-samples were collected in April–June 2015 to preclude intense winter emissions of PM and PAH from domestic coal-fired stoves (especially in Gliwice) and from heating plants, coal-fired as well. In whole Poland, the physicochemical properties of PM in winter (heating season) are entirely different from those in the rest of the year (Rogula-Kozłowska et al., 2013, 2016), probably because the PM from heating, moving with air masses, reaches even sites very remote from big PM sources (Rogula-Kozłowska et al., 2014). Also, the high ambient temperatures, insolation, and ozone concentrations of a hot summer period (July–August), favorable to the intense PAH transformations, and to the evaporation of light PAH from PM and their staying in gaseous form in the air (Srogi, 2007; Ravindra et al., 2008; Dvorská et al., 2011) were avoided. The air temperature in the measuring period was between 6.9–19.7°C (av. 14.9°C) in Gliwice and 7.5–23.3°C (av. 13.5°C) in Warsaw.

The 24-hr samples of PM_1 were collected simultaneously in the teaching rooms and their outsides, and simultaneously in Warsaw and in Gliwice. Both the rooms are located on the 3rd floor (approximately 8 to 10 m above the ground level). Neither air conditioning nor air cleaning equipment was installed in the rooms. They were heated with central heating radiators, there were no indoor PAH sources like smoking, cooking, etc. in the rooms. The tight PVC windows were opened only for airing before class and in the evenings during cleaning. Each room had a floor area of about 100 m², usually there were 20–30 people in it during class. The PM samplers were located in the room corners, about 2 m from the nearest wall, 5 (Gliwice) and 8 m (Warsaw) away from the windows and 6 (Gliwice) and 10 m (Warsaw) from the door.

All the outdoor samples were taken about 5 m above the ground level. The outdoor samplers were located 50 (Gliwice) and 70 m (Warsaw) – in a straight line – from the indoor samplers. In Warsaw, the distance to the nearest residential buildings, where coal and biomass might be combusted for heating, was 850 m; it was 350 m in Gliwice. The nearest coal-fired power stations were about 4 km away in Warsaw and 2.5 km in Gliwice. In European urban areas, an important source of PAH is road traffic (Ravindra et al., 2008). Both outdoor samplers were located about 200 m from busy roads. The samples were taken from Monday through Friday, when classes or lectures were held in the rooms. Altogether, 30 pairs of 24-hr samples of PM_1 were taken in each Gliwice and Warsaw (120 in total).

At each measuring site PM$_1$ was sampled using two identical samplers, two Atmoservice MVS in Warsaw and two Leckel KFG LVS in Gliwice, all equipped with identical jet impactors removing particles greater than 1 μm from the air stream (TSI Inc.). PM$_1$ was sampled at an air flow rate of 2.3 m^3/hr onto 47-mm QMA quartz fiber filters (Whatman). Before the exposure, the clean filters were heated at 650°C for 2 hr, conditioned in a weighing room (48 hr; relative air humidity 45% ± 5%; air temperature 20 ± 2°C) and weighed twice (with a 24 hr interval between weighings) on a Mettler Toledo AT microbalance (resolution of 2 μg) equipped with a Haug U-ionizer. The procedures for conditioning, weighing, storage, and transport of the samples and for the blank sample preparation complied with the QA/QC procedures of the reference method for gravimetric measurements (EN 12341:2014). The weighing accuracy, *i.e.*, a triple standard deviation from the mean of ten weighings of a blank filter (conditioned for 48 hr), was 20.5 μg.

The weighed filters were kept in tightly closed (sealed) containers in a refrigerator until their exposure. After the exposure and weighing, the filters with PM were put into containers, then tightly closed and enveloped in aluminum foil, and kept in a refrigerator (2–4°C) until the analysis.

The concentrations of PM$_1$-bound all: naphthalene (Na), acenaphthylene (Acy), acenaphthene (Ace), fluorene (Flu), phenantrene (Ph), anthracene (An), fluoranthene (Fl), pyrene (Py), benzo[a]anthracene (BaA), chrysene (Ch), benzo[b]fluoranthene (BbF), benzo[k]fluoranthene (BkF), benzo[a]pyrene (BaP), indeno[1,2,3-cd]pyrene (IP), dibenzo[ah]anthracene (DBA) and benzo[ghi]perylene (BghiP) were determined by chromatographically analyzing the dichloromethane extracts of the PM$_1$ samples (122 in total). The extraction, the chromatography technique, and validation parameters are described in detail in (Rogula-Kozłowska et al., 2013; Kozielska et al., 2015).

The cumulative health hazard from a mixture of PAH can be assessed by using the CEQ, MEQ, or TEQ equivalents relative to the carcinogenicity or mutagenicity of BaP, or to the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin, respectively. For each site and for indoor and outdoor PM$_1$-bound PAH, CEQ, MEQ, and TEQ were computed as linear combinations of the concentrations the PAH (square brackets) and their toxicity equivalence factors TEF (CEQ, Eq. (1)), PAH minimum mutagenic concentrations MMC (MEQ, Eq. (2)), and TCDD-TEF (TEQ, Eq. (3)). The values of TEF, MMC and TCDD-TEF for the PAH, used in Eqs. (1)–(3), are taken from Nisbet and LaGoy (1992), Durant et al. (1996), and Willett et al. (1997).

$$CEQ = 0.001 \times ([Acy] + [Ace] + [Flu] + [Ph] + [Fl] + [Py]) + 0.01 \times ([An] + [Ch] + [BghiP]) + 0.1 \times ([Na] + [BaA] + [BbF] + [BkF] + [IP]) + 1 \times [BaP] + 5 \times [DBA] \quad (1)$$

$$MEQ = 0.00056 \times [Acy] + 0.082 \times [BaA] + 0.017 \times [Ch] + 0.25 \times [BbF] + 0.11 \times [BkF] + 1 \times [BaP] + 0.31 \times [IP] + 0.29 \times [DBA] + 0.19 \times [BghiP] \quad (2)$$

$$TEQ = 0.000025 \times [BaA] + 0.00020 \times [Ch] + 0.000354 \times [BaP] + 0.00110 \times [IP] + 0.00203 \times [DBA] + 0.00253 \times [BbF] + 0.00487 \times [BkF] \quad (3)$$

The 7 US EPA carcinogenic PAH are: BaA, BaP, BbF, BkF, Ch, DBA, and IP. The share of the carcinogenic PAH (ΣPAH$_{carc}$) in ΣPAH (ΣPAH$_{carc}$/ΣPAH; US EPA, 2008) is determined as:

$$[\Sigma PAH_{carc}]/[\Sigma PAH] = ([BaA] + [BaP] + [BbF] + [BkF] + [Ch] + [DBA] + [IP]) \\ /([Na] + [Acy] + [Ace] + [Flu] + [Ph] + [An] + [Fl] \\ + [Py] + [BaA] + [Ch] + [BbF] + [BkF] + [BaP] + [DBA] \\ + [BghiP] + [IP]) \quad (4)$$

The factors computed for Warsaw and Gliwice were compared to those computed for other locations from the data taken from Oliveira et al. (2016), Mohammed et al. (2016), Zhu et al. (2015) and Hassanvand et al. (2015).

To determine the origin of the PM$_1$-bound PAH at the two sites, molecular diagnostic ratios (MDR) were computed and analyzed. The diagnostic ratios are mutual proportions between the ambient concentrations of single PAH or groups of PAH that have similar physicochemical properties (Tobiszewski and Namieśnik, 2012). The MDR analysis consists in comparison of these proportions for various PAH sources (car exhaust, flue gases from industrial processes, power stations, coal ovens, *etc*.). Although commonly applied in the PM-bound PAH source attribution, MDR must be used cautiously because the proportions are sensitive to the atmospheric conditions and can be the same for various PAH sources (Dvorská et al., 2011; Křůmal et al., 2013). To support the MDR analysis, principal component analysis (PCA) was applied to two 16 × 30 (Gliwice, indoor and outdoor) and two 15 × 30 (Warsaw, only 15 PAH - Na was not found in the PM samples) matrices of 24-hr PM$_1$-bound concentrations. The principal components with eigenvalues > 1.0 were chosen (Kaiser criterion). All the calculations were done using Statistica 8.0 software (StatSoft).

2. Results and discussion

The outdoor and indoor average PM$_1$-bound PAH concentrations in Warsaw and Gliwice in April–June 2015 were low compared to averages computed for other Polish cities in longer periods containing winters, and even several times lower than averages computed for winters alone; they resembled rather summer ones (Table 1, Fig. 1) (Rogula-Kozłowska et al., 2013; Rogula-Kozłowska, 2016; Kozielska et al., 2015). This agrees with numerous papers claiming that the energy production for heating is the main source of fine PM-bound PAH in Poland. The strong relation between combustion of coal for heat and power production (in summer mainly power) and the levels of ambient concentrations of PAH, especially BaP, in Poland, is also confirmed in the present work. The average concentrations of PM$_1$-bound BaP in April–June 2015 were 1.86 and 3.35 ng/m^3 in Warsaw and Gliwice, respectively. They were higher than in other European cities (Rogula-Kozłowska, 2015; GIOŚ, 2016). Some MDR indicate the predominant role of the combustion of coal or/and biomass in shaping the PM$_1$-bound PAH concentrations in Warsaw and Gliwice in the measuring period (Table 2) (Ravindra et al., 2008; Tobiszewski and Namieśnik, 2012; Rogula-Kozłowska, 2015).

The emissions from road traffic were also identified. The evidence in support of the contribution of the Diesel emissions

Table 1 – Concentrations of 16 PAH (ng/m³) in the air of the teaching rooms (indoor) and outside the buildings (outdoor) in Warsaw and Gliwice in spring 2015.

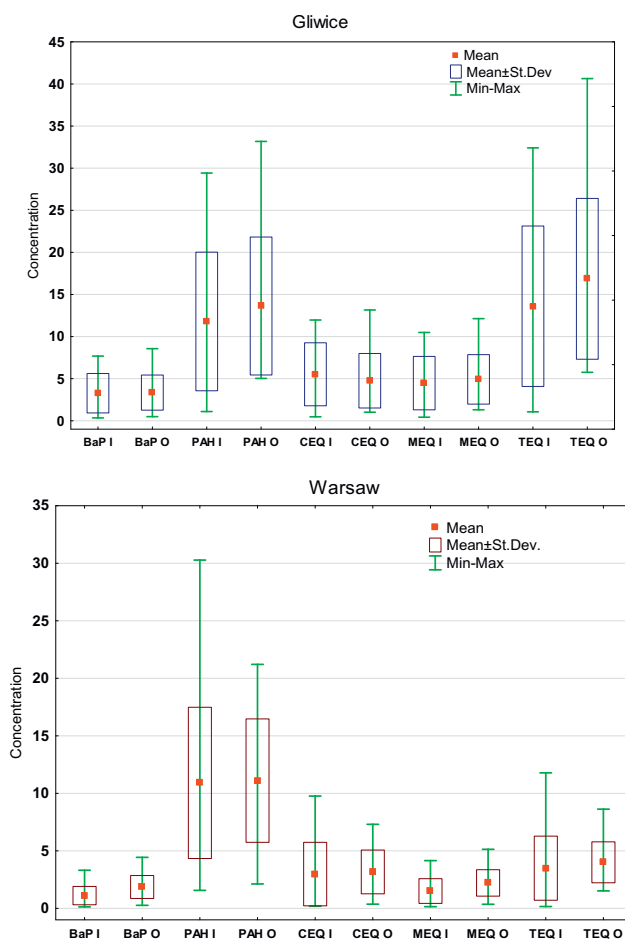|	Warszawa				Gliwice			
	Indoor		Outdoor		Indoor		Outdoor	
	Mean	St dev.	Mean	St dev.	Mean	St dev.	Mean	St dev.
Na	0.01	0.00	0.01	0.00	*0.03	0.03	*0.18	0.17
Acy	0.39	0.43	0.66	0.71	0.46	0.43	0.63	0.54
Ace	*0.10	0.11	*0.28	0.33	0.11	0.16	0.18	0.27
Flu	*0.77**	0.78	*0.30**	0.17	2.24**	2.34	1.53**	1.44
Ph	*0.61**	0.37	*0.17**	0.11	0.69**	1.17	0.52	0.60
An	0.37**	0.29	0.42	0.29	*2.00**	2.65	*0.42**	0.44
Fl	3.71	3.09	3.69**	2.34	3.03	2.46	2.47**	2.09
Py	*0.45	0.27	*0.26**	0.26	0.52	0.38	0.86**	1.03
BaA	0.63**	0.53	0.74**	0.53	2.12**	1.56	2.71	2.09
Ch	1.49**	1.77	1.56	1.13	2.49**	2.02	2.33	2.27
BbF	0.42**	0.33	0.40**	0.21	1.73**	1.24	2.17**	1.14
BkF	*0.15**	0.18	*0.28**	0.12	1.28**	0.92	1.64**	0.94
BaP	*1.11**	0.79	*1.86**	1.00	3.27**	2.34	3.35**	2.09
IP	0.27	0.22	0.18**	0.15	*0.58	0.85	*1.32**	1.04
DBA	0.34	0.39	0.22	0.25	0.32	0.44	0.12	0.16
BghiP	0.08**	0.16	0.09**	0.13	0.73**	0.72	0.72**	0.53

PAH: polycyclic aromatic hydrocarbons; Na: naphthalene; Acy: acenaphthylene; Ace: acenaphthene; Flu: fluorene; Ph: phenanthrene; An: anthracene; Fl: fluoranthene; Py: pyrene; BaA: benzo[a]anthracene; Ch: chrysene; BbF: benzo[b]fluoranthene; BkF: benzo[k]fluoranthene; BaP: benzo[a]pyrene; IP: indeno[1,2,3-cd]pyrene; DBA: dibenzo[ah]anthracene; BghiP: benzo[ghi]perylene.

* PAH whose indoor and outdoor concentrations at a site are statistically significantly different.

** PAH whose indoor or outdoor concentrations at one site are statistically significantly different from those at the other site (both according to the Mann–Whitney U test; $\alpha_{assump.} = 0.05$, $p_{comp.} < 0.05$).

Fig. 1 – Ranges of the concentrations of BaP, ΣPAH (ng/m³), and of the values of CEQ, MEQ (ng/m³), and TEQ (pg/m³) in the air of the teaching rooms (I) and outside the buildings (O) in Warsaw and Gliwice in spring 2015. PAH: polycyclic aromatic hydrocarbons; CEQ: carcinogenic equivalent; MEQ: mutagenic equivalent; TEQ: TCDD-toxic equivalent.

was the [Fl]/([Fl] + [Py]) ratios higher than 0.5 (Tobiszewski and Namieśnik, 2012). The MDR for some PAH were ambiguous ([BaA]/([Ch] + [BaA]), [BaA]/[BaP]), and it was hard to tell from them which source, coal and biomass combustion or combustion of liquid fuels in car engines, had greater effect on the concentrations of these PAH. In the circumstances of many source contribution to PM concentrations, MDR can be of a limited value (Galarneau, 2008). It is improbable that traffic emissions have no significant effect on ambient PAH concentrations in great cities such as Warsaw and Gliwice, this effect is rather much less than that of solid fuel combustion.

The conclusions drawn from the MDR analysis refer only to the PM₁-bound parts of ambient PAH. Two- or three-ring PAH can only be vaporous in the air, four-ring PAH can change their phase depending on ambient conditions, and only five- or more ring ambient PAH occur always in the PM-bound phase (Ravindra et al., 2008). In a highway tunnel, two-ring PAH can make 76% of total PAH in the air, in diesel and gasoline engine exhaust the sum of two- and three-ring PAH can make more than 60 and 70%, respectively (Khalili et al., 1995). In the 24-hr samples from Warsaw, the PM₁-bound naphthalene was not found at all; it was found only in several samples from Gliwice. Therefore, the correct determination of ambient PAH sources should rely on more comprehensive analysis including both gaseous and particulate PAH

concentrations, and it is obvious that the presented here results can give only a rough idea of the PM₁-bound PAH origins. It is highly probable that including gaseous PAH into the analysis would give another source attribution of ambient PAH at both sites. This is also confirmed by the results of principal component analysis (PCA) (Table 3).

At both sites, the majority of the PAH were correlated with the first principal component (PC1), therefore identifying a dominant source with PC1 was impossible (Thurston and Spengler, 1985). However, PCA revealed some differences between the two sites. In Warsaw, the PAH commonly linked to road traffic emissions (e.g., Flu, Ph, BghiP, and IP) were correlated stronger with PC1, in Gliwice it was PAH linked to coal and biomass combustion (Fl, Py, BaA, Ch, BkF, BbF; Harrison et al., 1996; Ravindra et al., 2008; Kozielska, 2015). In Warsaw, the traffic emission markers (BghiP, DBA, and Ph) were correlated with PC2, in Gliwice - the markers of coal combustion (Ch and Py) (Kozielska, 2015). This means that the effect of road traffic on PM₁-bound PAH concentrations prevails in Warsaw and it is drowned out by combustion of coal and biomass in Gliwice.

Table 2 – Molecular diagnostic ratios (MDR) for PM$_1$-bound PAH in the air of the teaching rooms (indoor) and outside the buildings (outdoor) in Warsaw and Gliwice in spring 2015.

MDR	Warszawa		Gliwice	
	Indoor	Outdoor	Indoor	Outdoor
[Ph]/([Ph + An]) [a]	0.63 (0.40–0.81)	0.34 (0.08–0.52)	0.24 (0.01–0.85)	0.48 (0.05–0.87)
[Fl]/([Py] + [Fl])	0.81 (0.42–0.94)	0.89 (0.55–0.99)	0.76 (0.49–0.94)	0.70 (0.50–0.91)
[BaA]/([Ch] + [BaA])	0.38 (0.19–0.69)	0.33 (0.19–0.52)	0.48 (0.27–0.92)	0.55 (0.26–0.79)
[BaA]/[BaP]	0.63 (0.22–1.36)	0.43 (0.19–1.07)	0.68 (0.39–0.93)	0.98 (0.24–3.76)
[IP]/([IP] + [BghiP])	0.75 (0.50–0.98)	0.66 (0.50–0.98)	0.38 (0.03–0.64)	0.58 (0.01–0.97)
[BaP]/[BghiP]	86.08 (3.47–331.53)	114.25 (5.98–443.77)	27.28 (2.67–226.57)	9.67 (0.75–43-98)
PAH$_{comb}$/∑PAH	0.75 (0.57–0.93)	0.82 (0.73–0.91)	0.70 (0.30–0.93)	0.81 (0.62–0.93)

PM: particulate matter; PAH: polycyclic aromatic hydrocarbons; Ph: phenantrene; An: anthracene; Fl: fluoranthene; Py: pyrene; BaA: benzo[a]anthracene; Ch: chrysene; BaP: benzo[a]pyrene; IP: indeno[1,2,3-cd]pyrene; BghiP: benzo[ghi]perylene.
[a] PAH diagnostic ratios for various sources adopted from Tables 3 and 4 in Rogula-Kozłowska (2015) and references cited therein.

In fact, in Poland, the fine PM behavior follows two patterns. The one followed by PM in southern Poland can be clearly seen in the results from Gliwice, the other, for the rest of Poland, is visible in the results from Warsaw. In the cities of southern Poland, coal and biomass combustion greatly, and industry to a lesser extent, affect the concentrations and the composition of fine PM (Rogula-Kozłowska et al., 2014, 2015, 2016; Rogula-Kozłowska, 2016). In Warsaw, a city located in central Poland, traffic emissions can contribute more than 15% to PM$_{2.5}$ concentrations even in a heating season (Majewski and Rogula-Kozłowska, 2016). The ambient concentrations of fine PM and its components (in the present work PAH, in others *e.g.*, metals) are higher in southern than in central Poland (Majewski and Rogula-Kozłowska, 2016; Rogula-Kozłowska,

2015; Fig. 1). The differences between the outdoor 24-hr concentrations of Flu, Fl, Py, BaP, BbF, BkF, BaP, IP, and BghiP in Warsaw and Gliwice in April–June 2015 are statistically significant (Mann–Whitney U test; $\alpha_{assump.}$ = 0.05, $p_{comp.}$ < 0.05), and all their means, except the one for Fl, are higher in Gliwice (Table 1). This higher PM$_1$-bound Fl mean concentration and, due to it, higher mass contribution of four-ring PAH to ∑PAH in Warsaw (Fig. 2) also reflect the road traffic effects in PM$_1$ (Caricchia et al., 1999; Kulkarni and Venkataraman, 2000).

The MDR analysis and PCA indicated the same sources for indoor and outdoor PM$_1$-bound PAH, both in Gliwice and Warsaw (Tables 2 and 3). Also, the mass distributions of outdoor and indoor PM$_1$-bound ∑PAH among three-, four-, five- and six-ring PAH were similar (Fig. 2).

Table 3 – Results from principal components analysis (PCA) done FOR PM$_1$-bound PAH in the air of the teaching rooms (indoor) and outside the buildings (outdoor) in Warsaw and Gliwice in spring 2015.

	Warszawa								Gliwice						
	Indoor			Outdoor					Indoor			Outdoor			
	PC1	PC2	PC3	PC1	PC2	PC3	PC4	PC5	PC1	PC2	PC3	PC1	PC2	PC3	PC4
Na	–	–	–	–	–	–	–	–	0.4	−0.2	−0.6	**0.9**	0.4	0.2	0.0
Acy	**−0.7** [a]	**−0.6**	0.3	**0.8**	0.0	−0.5	−0.3	0.1	**−0.8**	**0.6**	−0.1	0.4	**0.7**	−0.5	−0.4
Ace	−0.4	**−0.8**	−0.1	**0.7**	0.5	0.4	0.0	0.0	**−0.6**	**−0.7**	0.2	0.0	0.3	**−0.8**	0.0
Flu	**−0.8**	0.3	−0.2	**0.8**	−0.3	0.4	0.0	0.0	**−0.8**	0.2	−0.3	0.3	**−0.8**	−0.4	−0.2
Ph	−0.3	**−0.8**	0.3	**0.7**	**−0.6**	0.0	0.4	0.1	−0.4	**0.7**	0.1	−0.2	−0.1	**−0.9**	0.0
An	−0.4	**−0.7**	0.2	0.1	**−0.6**	0.5	−0.1	−0.2	0.4	−0.1	**−0.7**	0.0	−0.5	0.2	**−0.6**
Fl	−0.4	0.4	**0.8**	0.5	−0.4	0.5	−0.4	−0.3	**−0.8**	−0.4	0.2	**0.9**	0.1	0.3	−0.1
Py	**−0.6**	−0.1	0.2	0.2	0.0	−0.2	**0.8**	**−0.6**	**−0.8**	−0.5	0.1	**0.6**	**−0.7**	−0.1	0.0
BaA	**−0.8**	0.5	0.0	**0.8**	0.3	−0.1	0.0	−0.5	**−0.9**	0.2	−0.1	**0.9**	0.3	0.2	0.0
Ch	**−0.6**	**0.7**	0.0	**0.9**	0.2	−0.2	−0.1	0.0	**−0.8**	**−0.6**	0.0	0.5	**−0.7**	0.0	0.4
BbF	**−0.9**	−0.1	0.0	**0.6**	−0.1	0.1	0.5	0.5	**−1.0**	−0.1	−0.1	**1.0**	−0.1	−0.2	0.1
BkF	**−0.8**	0.4	0.4	**0.9**	−0.1	−0.1	0.2	0.2	**−1.0**	−0.1	−0.2	**0.9**	−0.1	−0.3	−0.1
BaP	**−0.8**	−0.4	−0.4	**0.9**	−0.3	−0.1	−0.1	0.2	**−1.0**	−0.2	0.0	**0.9**	0.1	0.1	0.0
IP	**−0.9**	0.0	−0.3	**0.8**	0.3	−0.2	−0.2	0.2	**−0.7**	0.4	−0.1	**0.9**	−0.2	0.0	0.1
DBA	**−0.8**	−0.4	−0.4	0.2	−0.5	**−0.7**	−0.3	−0.3	−0.2	**0.6**	0.5	**0.9**	0.3	0.1	−0.2
BghiP	**−0.6**	**0.8**	−0.1	**0.6**	**0.6**	0.4	−0.1	−0.2	**−0.8**	0.3	−0.2	0.3	0.4	0.0	**0.7**
Eigenvalue	7.1	4.3	1.4	7.0	2.2	1.9	1.4	1.2	8.8	2.8	1.5	7.5	3.1	2.3	1.2
Variance	0.5	0.3	0.1	0.5	0.1	0.1	0.1	0.1	0.6	0.2	0.1	0.5	0.2	0.1	0.1

Na: naphthalene; Acy: acenaphthylene; Ace: acenaphthene; Flu: fluorene; Ph: phenantrene; An: anthracene; Fl: fluoranthene; Py: pyrene; BaA: benzo[a]anthracene; Ch: chrysene; BbF: benzo[b]fluoranthene; BkF: benzo[k]fluoranthene; BaP: benzo[a]pyrene; IP: indeno[1,2,3-cd]pyrene; DBA: dibenzo[ah]anthracene; BghiP: benzo[ghi]perylene.
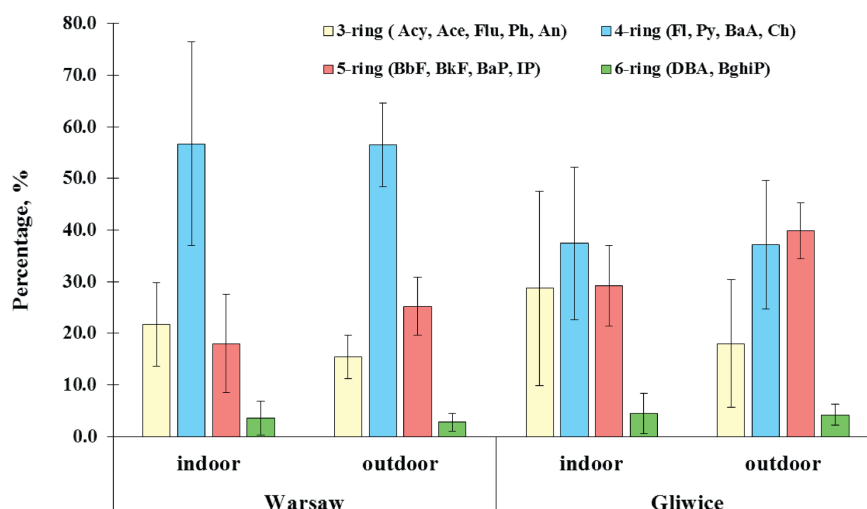[a] Loadings whose absolute values are greater than 0.6 are in bold.

Fig. 2 – Mass percentages (arithmetic mean (bar), and standard deviation (whiskers)) of three-, four-, five- and six-ring PAH in ΣPAH in the air of the teaching rooms (indoor) and outside the buildings (outdoor) in Warsaw and Gliwice in spring 2015. PAH: polycyclic aromatic hydrocarbons.

This affinity between indoor and outdoor PM_1-bound PAH was due to the penetration of PM_1-bound PAH from outdoors to indoors. In both cities (rooms), the indoor and outdoor 24-hr concentrations of PM_1 were correlated (Fig. 3), meaning their concerted variations and strong effect of outdoor on indoor PM_1. Despite this, statistically significant differences between the indoor and outdoor 24-hr concentrations were found for 5 PAH in Warsaw and 3 PAH in Gliwice (Mann–Whitney U test; $\alpha_{assump.} = 0.05$, $p_{comp.} < 0.05$; Table 1). However, in Gliwice, where the average I/O for ΣPAH was 1 and its 24-hr values were between 0.4 and 2.2 (Fig. 3), it neither appeared in the proportions I/O for these 3 PAH nor for total PAH (ΣPAH). In Warsaw, although the average concentrations of ΣPAH outdoors and indoors were close, its average I/O was almost 1.5 and the 24 hr values of I/O were from 0.1 to 7.5. Much higher indoor than outdoor concentrations were noted
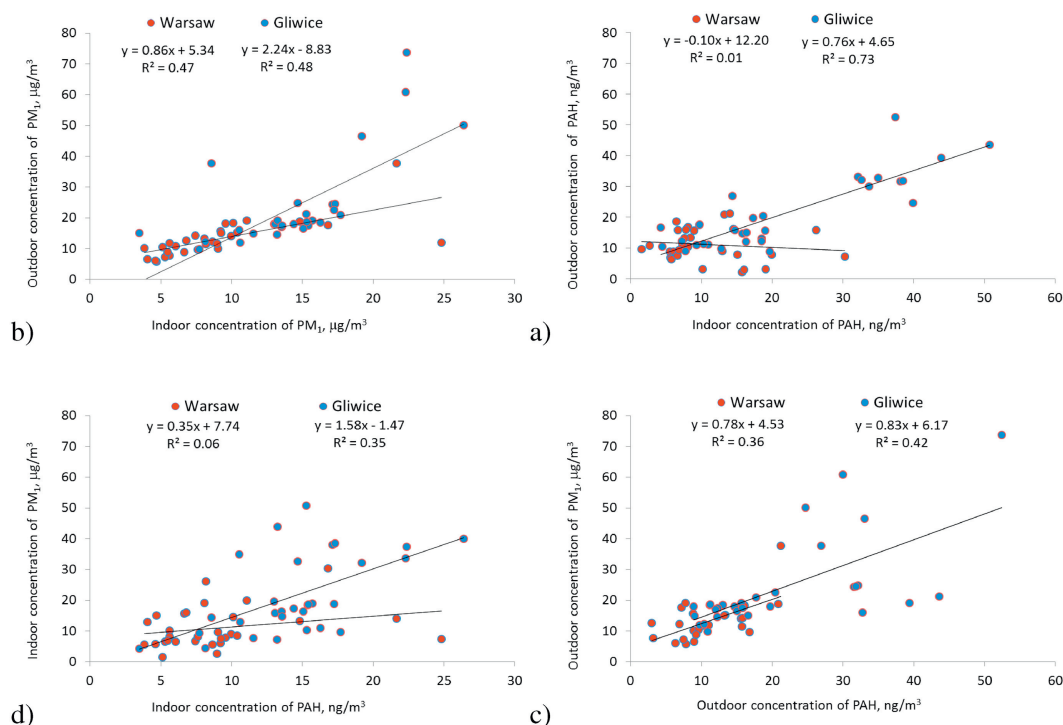

Fig. 3 – Linear correlation between indoor (a) and outdoor (b) 24-hr concentrations of PM_1 and PM_1-bound PAH, and between 24-hr concentrations of PM_1 and PM_1-bound PAH indoor (c) and outdoor (d). PAH: polycyclic aromatic hydrocarbons; PM: particulate matter.

for Flu, Ph, and Py in Warsaw, and mainly these three PAH made the 24 hr ΣPAH I/O be often much greater than 1. In Warsaw, Ph was strongly correlated with PC3 (Table 3), probably meaning an indoor PAH source. Both the sites (rooms) were chosen so that they were not affected by typical PAH sources, but the existence of an unidentified ones cannot be precluded. Schober et al. (2014) showed that after a 2-hr e-cigarette vaping session the concentration of Fen in a room in an office building was the highest among the concentrations of 15 PAH they investigated. Flu, Ph, and Py can also be emitted by warm chip encapsulations in working personal computers, and blown out of the cases by cooling fans as gases or bound to PM (Ren et al., 2006). These indoor provenance PAH could have also come from laboratories. Wherever they came from, their gaseous parts adhered to PM_1 coming from outdoors. This is why the 24-hr indoor and outdoor concentrations of ΣPAH in Warsaw are not correlated (Fig. 3). Neither were 24-hr indoor concentrations of PM_1 and ΣPAH in Warsaw correlated, while outdoor ones were (Fig. 3). The greater part of indoor gaseous PAH came from outdoors in both Warsaw and Gliwice. Maybe in Warsaw, where rather the road traffic effects prevail in atmospheric PAH (Majewski and Rogula-Kozłowska, 2016), the light gaseous PAH make much greater part in ΣPAH than they do in Gliwice, both outdoors and indoors. The bounded volume of the rooms, stable air temperature (about 20°C), calm air, lack of direct insolation, *etc.* are favorable for sorption of volatile and semi-volatile compounds onto PM (Harrison et al., 1996; Srogi, 2007; Ravindra et al., 2008; Dvorská et al., 2011). The mass contributions of three-ring PAH to ΣPAH, greater indoors than outdoors at both sites, confirm this (Fig. 2).

CEQ, MEQ, TEQ, and to the lesser extent [ΣPAH$_{carc}$]/[ΣPAH] (Eqs. (1)–(4)) estimate the toxicity (carcinogenicity) and health hazard from mixtures of PAH considered a single substance (Fig. 4).
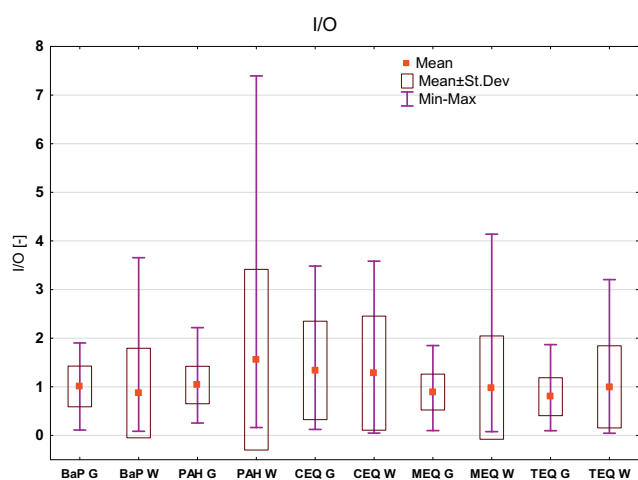
BaA, BaP, BbF, BkF, Ch, DBA, and IP are considered carcinogens (US EPA, 2008). The carcinogenic potential of ΣPAH in humans can be expressed roughly as the share of their total (ΣPAH$_{carc}$) in ΣPAH, *i.e.*, as [ΣPAH$_{carc}$]/[ΣPAH] (Eq. (4)). However, this proportion is not very reliable as a toxicity estimate because it does not factor in the circumstances at the site where the ΣPAH is measured (*e.g.*, its ambient concentrations). It can give some idea of the potential carcinogenicity of the PM_1-bound ΣPAH, but, for example, it allows for considering the PM_1-bound ΣPAH in two Portugal preschools (potentially) more carcinogenic than those in the two Polish cities, and even in Asia where ΣPAH concentrations are really vast (Table 4), while the carcinogenic hazard from PM_1-bound ΣPAH to humans in Portugal is probably lowest among all these sites because of very low ΣPAH concentrations. This coefficient, averaged, indicates that the PM_1-bound ΣPAH in Gliwice had higher carcinogenic potential than in Warsaw, equally outdoors and indoors, and the indoor one was lower than the outdoor one in both cities.

CEQ seems to indicate the PM_1-bound ΣPAH carcinogenicity more adequately because it uses TEF, which relativize the particular PAH carcinogenicities to the BaP carcinogenicity and its computing involves all the 16 PAH (Eq. (1)). But again, although the PM_1-bound ΣPAH and BaP concentrations (indoors and outdoors) in two preschools in Portugal were many times lower than in Warsaw and Gliwice, the CEQ in Poland and Portugal are comparable (Table 4). The closeness of the CEQ in Poland and in Portugal, despite several times higher concentrations in Poland than in Portugal, is due to low heavy PAH content of PM_1-bound ΣPAH in the Polish cities compared to the localities in Portugal; it is also lower than in other European localities (Rogula-Kozłowska, 2015). Five- and six-ring PAH (especially DBA) have very high TEF (Eq. (1)). But the concentrations of PM_1-bound ΣPAH, including BaP, in Polish cities in a non-heating period are among the highest in Europe (Rogula-Kozłowska, 2015), they often are as high as in polluted Asian cities, and the PM_1-bound ΣPAH in Warsaw and Gliwice probably cause a greater carcinogenic hazard to humans than those in Portugal (Table 4). In both Warsaw and Gliwice, the indoor and outdoor average CEQ were comparable (in Gliwice the indoor one was slightly higher). In other locations indoor and outdoor CEQ also were close to one another (Table 4).

In this way, using equations similar to the Eqs. (1)–(3), other factors estimating the toxicity of the 16 PAH mixture can be defined. Such a definition needs selecting a reference chemical, whose toxicity is well recognized, and determining the toxicity equivalency factors (equation coefficients) for all the 16 PAH relative to it. The term "toxicity" stands for any kind of harmfulness of the PAH mixture. The factors for the PAH considered non-harmful are set equal to zero.

MEQ is defined relative to the BaP mutagenicity (Eq. (2); MMC, Durant et al., 1996), and TEQ relative to the ability of 2,3,7,8-tetrachlorodibenzo-p-dioxin to suppress the enzymatic activity of 7-etoksyresorufin O-deetylase (EROD) in fish liver cells (Eq. (3); TCDD-TEF; Willett et al., 1997).

The indoor MEQ and TEQ were lower than the outdoor ones everywhere, in the Polish and in all the exemplary localities (Table 4).

Therefore, in public utility buildings, such as universities, preschools, business offices, *etc.*, where PAH come from outdoors

Fig. 4 – Ranges of the indoor/outdoor proportions (I/O) of the concentrations of BaP, ΣPAH (PAH), CEQ, MEQ, and TEQ in Gliwice (G) and Warsaw (W). PAH: polycyclic aromatic hydrocarbons; CEQ: carcinogenic equivalent; MEQ: mutagenic equivalent; TEQ: TCDD-toxic equivalent; BaP: benzo[a]pyrene.

Table 4 – Carcinogenic equivalent (CEQ), mutagenic equivalent (MEQ), TCDD-toxic equivalent (TEQ), and the share of carcinogenic PAH in ΣPAH (ΣPAH$_{carc}$/ΣPAH) for PM$_1$-bound PAH in indoor and outdoor air in Warsaw, Gliwice, and other localities.

Location		Sampling period	Fraction		ΣPAH (n)*	BaP	CEQ	MEQ	TEQ	PAH$_{canc}$/ΣPAH	Reference
					ng/m^3				pg/m^3	–	
North of Portugal	Preschool 1	IV–VI 2013	indoor	PM$_1$	0.86 (18)	0.03	1.61	0.19	1.08	0.66	Oliveira et al. (2016)
			outdoor		1.40 (18)	0.05	2.45	0.29	1.69	0.70	
			indoor	PM$_{2.5}$	1.45 (18)	0.05	2.94	0.35	1.87	0.65	
			outdoor		1.62 (18)	0.07	2.49	0.34	1.92	0.68	
	Preschool 2		indoor	PM$_1$	3.11 (18)	0.12	3.57	0.48	2.61	0.46	
			outdoor		2.52 (18)	0.12	2.69	0.42	2.20	0.52	
			indoor	PM$_{2.5}$	4.23 (18)	0.26	4.77	0.79	3.67	0.50	
			outdoor		4.55 (18)	0.30	5.76	0.93	4.64	0.58	
Harbin, China	Residential buildings	Annual	indoor	PM$_{2.5}$	42.2 (16)	3.10	7.88	6.61	26.69	0.43	Mohammed et al. (2016)
			outdoor		141.20 (16)	8.10	19.36	16.95	69.26	0.38	
Jinan, China	Business office	Summer 2010	indoor	PM$_{2.5}$	29.20 (15)	0.40	1.16	1.61	11.82	0.29	Zhu et al. (2015)
			outdoor		30.78 (15)	0.99	2.17	3.19	24.54	0.40	
		Autumn 2010	indoor		111.07 (15)	5.60	14.13	15.99	124.95	0.55	
			outdoor		140.34 (15)	10.66	21.49	25.79	176.86	0.67	
Tehran, Iran	Retirement home	V 2012–V 2013	indoor	PM$_1$	242.05 (16)	5.11	77.49	20.56	146.69	0.47	Hassanvand et al. (2015)
			outdoor		283.52 (16)	5.66	89.09	23.62	170.84	0.47	
			indoor	PM$_{2.5}$	304.59 (16)	5.80	87.78	24.11	176.29	0.44	
			outdoor		352.07 (16)	6.51	99.45	27.05	196.23	0.43	
			indoor	PM$_{10}$	350.24 (16)	6.44	90.49	26.59	193.26	0.41	
			outdoor		425.56 (16)	7.50	111.31	31.13	226.60	0.40	
	School dormitory		indoor	PM$_1$	222.85 (16)	4.76	75.89	19.32	136.21	0.47	
			outdoor		288.98 (16)	5.88	96.75	24.30	174.84	0.47	
			indoor	PM$_{2.5}$	281.27 (16)	5.42	81.24	22.04	160.35	0.43	
			outdoor		361.74 (16)	6.73	102.61	27.49	198.73	0.42	
			indoor	PM$_{10}$	317.43 (16)	5.82	86.61	24.33	179.76	0.41	
			outdoor		429.62 (16)	7.54	94.56	31.23	224.42	0.39	
Warsaw, Poland	University teaching room	IV–VI 2015	indoor	PM$_1$	10.90 (16)	1.11	2.98	1.48	3.50	0.38	This study
			outdoor		11.09 (16)	1.86	3.15	2.21	4.00	0.48	
Gliwice, Poland			indoor		21.61 (16)	3.27	5.53	4.48	13.61	0.52	
			outdoor		21.14 (16)	3.35	4.72	4.91	16.85	0.63	

(n)*—number of PAH in the mixture.

and no indoor PAH sources exist, PM$_1$-bound PAH do not cause greater carcinogenic hazard than those outdoors. Slightly higher PM$_1$-bound PAH concentrations can occur indoors because sorption of gaseous PAH to PM is more effective indoors than outdoors, but rather 2-, 3-, and 4-ring PAH occur as gases in ambient air, and they are not considered carcinogens or mutagens.

3. Conclusions

In two big cities, Warsaw and Gliwice (Poland), the 24-hr concentrations of PM$_1$ and 16 PAH bound to PM$_1$ were determined in two university teaching rooms and in atmospheric air in their outsides. The results were used to asses and compare indoor and outdoor health hazards from PM$_1$-bound PAH at the two sites.

The indoor PM$_1$ concentrations were close to the outdoor ones at both sites, and so were the ΣPAH concentrations. The indoor PM$_1$ and ΣPAH concentration averages were 14.6 μg/m^3 and 21.6 ng/m^3, respectively, in Gliwice and 9.0 μg/m^3 and 10.9 ng/m^3 in Warsaw. The outdoor ones were 23.9 μg/m^3 and 21.1 ng/m^3 in Gliwice and 13.2 μg/m^3 and 11.1 ng/m^3 in Warsaw.

Moreover, the indoor and the outdoor concentrations of the majority of the 16 PM$_1$-bound PAH were very close, both in Warsaw and Gliwice. It means that the balance between indoor and outdoor PM$_1$-bound PAH is maintained and the atmosphere is the indoor PAH source. The carcinogenicity and mutagenicity of the 16 PM$_1$-bound PAH mixture indoors were also close to those outdoors, both in Warsaw and Gliwice. The health hazard from the 16 US EPA PM$_1$-bound PAH seems not to be greater in teaching rooms than outside and to be controlled by outside pollution conditions unless indoor pollution sources exist. There were no sources of PAH in the two examined teaching rooms in Warsaw and Gliwice, but the indoor concentrations of phenanthrene, fluorene, and pyrene in the Warsaw teaching room were higher than the outdoor ones. Most probably, in Warsaw, outside the room, some unidentified source of gaseous form of these PAH existed in the building. These PAH may have come from electronic cigarettes or hot parts of working personal computers.

Nevertheless, the PM$_1$-bound PAH concentrations in teaching rooms and the health hazard from them are at least strongly affected by outdoor (ambient) PM$_1$ and PAH concentrations and conditions, but in fact it rather must be assumed that in most cases the indoor concentrations are directly determined by the outdoor ones. In Gliwice, the greater part of

the PAH emissions is due to coal and biomass combustion, therefore PM_1-bound PAH contain much of heavy PAH, including BaP, and the indoor and outdoor health hazards from their mixture are greater than in Warsaw, where PAH come mainly from road traffic. The ambient 2-, 3-, and 4- ring PAH can occur in gaseous phase at higher and bind to PM_1 at lower ambient temperatures, but they do not affect the toxicity of the PM_1-bound PAH mixture significantly.

Acknowledgments

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